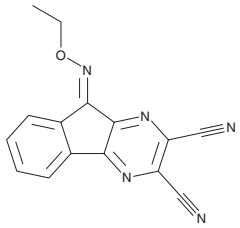
PRODUCT INFORMATION

DUBs-IN-2

Item No. 36229

CAS Registry No.: 924296-19-5
Formal Name: 9-(ethoxyimino)-9H-indeno[1,2-b] pyrazine-2,3-dicarbonitrile
Synonyms: Deubiquitinase Inhibitor 2, DUBs Inhibitor 2, HY-50737A
MF: $C_{15}H_9N_5O$
FW: 275.3
Purity: ≥95% (mixture of cis/trans isomers)
UV/Vis.: λ_{max}: 211, 226, 278, 322 nm
Supplied as: A solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

DUBs-IN-2 is supplied as a solid. A stock solution may be made by dissolving the DUBs-IN-2 in the solvent of choice, which should be purged with an inert gas. DUBs-IN-2 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of DUBs-IN-2 in these solvents is approximately 5 mg/ml.

Description

DUBs-IN-2 is an inhibitor of ubiquitin-specific protease 8 (USP8; IC_{50} = 280 nM).[1] It is selective for USP8 over USP7 (IC_{50} = >100 µM). DUBs-IN-2 increases the level of programmed cell death protein ligand 1 (PD-L1) in H460 cells when used at a concentration of 2 µM and PC-9 cells at 2 and 4 µM.[2] Intraperitoneal injection of DUBs-IN-2 (1 mg/kg every other day), in combination with an anti-PD-L1 antibody and paclitaxel (Item No. 10461), decreases tumor weight, tumor size, and lung metastasis and increases $CD8^+$ T cell numbers within the primary tumor in a 4T1 murine mammary carcinoma model.[3]

References

1. Colombo, M., Vallese, S., Peretto, I., *et al.* Synthesis and biological evaluation of 9-oxo-9H-indeno [1,2-*b*]pyrazine-2,3-dicarbonitrile analogues as potential inhibitors of deubiquitinating enzymes. *ChemMedChem* **5(4)**, 552-558 (2010).
2. Xiong, W., Gao, X., Zhang, T., *et al.* USP8 inhibition reshapes an inflamed tumor microenvironment that potentiates the immunotherapy. *Nat. Commun.* **13(1)**, 1700 (2022).
3. Xie, F., Zhou, X., Li, H., *et al.* USP8 promotes cancer progression and extracellular vesicle-mediated CD8+ T cell exhaustion by deubiquitinating the TGF-β receptor TβRII. *EMBO J.* **41(16)**, e108791 (2022).

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